

L Number	Hits	Search Text	DB	Time stamp
1	2	6077826.pn.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/06/11 08:57
2	2	wo adj "9726905"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/06/11 09:14
3	21	peptide same water same flux	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/06/11 09:15

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NEWS	6	Mar 08	Gene Names now available in BIOSIS
NEWS	7	Mar 22	TOXLIT no longer available
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NEWS	16	Apr 22	Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
NEWS	17	Apr 22	BIOSIS Gene Names now available in TOXCENTER
NEWS	18	Apr 22	Federal Research in Progress (FEDRIP) now available
NEWS	19	Jun 03	New e-mail delivery for search results now available
NEWS	20	Jun 10	MEDLINE Reload
NEWS	21	Jun 10	PCTFULL has been reloaded
NEWS EXPRESS			February 1 CURRENT WINDOWS VERSION IS V6.0d, CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP), AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
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=> s water (s) peptide (s) flux

L1 242 WATER (S) PEPTIDE (S) FLUX

=> s water (s) peptide (s) flux (s) epithel?

L2 33 WATER (S) PEPTIDE (S) FLUX (S) EPITHEL?

=> dup rem l2

PROCESSING COMPLETED FOR L2

L3 15 DUP REM L2 (18 DUPLICATES REMOVED)

=> d l3 total ibib kwic

L3 ANSWER 1 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 2001:158728 BIOSIS

DOCUMENT NUMBER: PREV200100158728

TITLE: Synthetic macromolecular channel assembly for transport of chloride ions through epithelium useful in treating cystic fibrosis.

AUTHOR(S): Tomich, John M. (1); Iwamoto, Takeo; Sullivan, Lawrence P.
CORPORATE SOURCE: (1) Manhattan, KS USA

ASSIGNEE: Kansas State University Research Foundation

PATENT INFORMATION: US 6077826 June 20, 2000

SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (June 20, 2000) Vol. 1235, No. 3, pp. No
Pagination. e-file.
ISSN: 0098-1133.

DOCUMENT TYPE: Patent

LANGUAGE: English

AB The present invention is directed to multiple-peptide channel assemblies for transport of anions such as chloride ions through epithelial cells, synthetic peptides capable of forming such channel assemblies and methods for using channel assemblies in therapeutic contexts for altering the flux of water across epithelial cells. The channel assemblies are composed of a plurality of peptides that transport through the membrane of an epithelial cell and provide for alteration of the

flux of water through the cell. The peptides are soluble in water to a level of at least 1 mM and exhibit at least about 50% helical content when dispersed in a 40% trifluoroethanol/60% water solution. The peptides ideally have the amino acid sequence ABC(X)_n DEF, where A, B, C, D, F and X are individual amino acid. . . from the group consisting of D, E, and F is a charged amino acid. The method hereof provides for altering flux of water from an epithelial cell and includes providing from 3-6 peptides capable of forming a channel assembly with each of such peptides having from about 18-30 amino acid residues therein, then contacting the peptides with a surface of an epithelial cell to cause the peptides to embed therein and alter the flux of water across the cell.

L3 ANSWER 2 OF 15 MEDLINE DUPLICATE 1
 ACCESSION NUMBER: 1999103935 MEDLINE
 DOCUMENT NUMBER: 99103935 PubMed ID: 9886985
 TITLE: Effect of sorbin on electrolyte transport in rat and human intestine.
 AUTHOR: Eto B; Boisset M; Griesmar B; Desjeux J F
 CORPORATE SOURCE: Conservatoire National des Arts et Metiers, Laboratoire de Biologie, 75141 Paris 03, France.
 SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY, (1999 Jan) 276 (1 Pt 1) G107-14.
 Journal code: 0370511. ISSN: 0002-9513.
 PUB. COUNTRY: United States
 Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199903
 ENTRY DATE: Entered STN: 19990324
 Last Updated on STN: 19990324
 Entered Medline: 19990309

AB Stimulating water absorption in the colon represents an important target to reduce stool output in secretory diarrhea. Recently,

a 153-amino-acid peptide was isolated from porcine upper small intestine and purified, taking into account the increase of water absorption in guinea pig gallbladder. Accordingly, this peptide was named sorbin. The aim of the present study was to determine if the COOH-terminal heptapeptide of sorbin (C7-sorbin) participates. . . or humans were mounted in Ussing chambers to measure the changes in short-circuit current (DeltaIsc) and net 22Na and 36Cl fluxes (JNanet and JClnet) after serosal exposure of 10⁻⁷ to 10⁻³ M C7-sorbin. In fasted rat intestine, C7-sorbin (10⁻⁴ M) induced. . . (10⁻³ M) inhibited the increase in Isc induced by a series of 10 secretory agents such as secretin, vasoactive intestinal peptide, PGE2, and serotonin. In HT-29-Cl19A cells, C7-sorbin induced an increase in Isc, with a maximal effect at 10⁻³ M (DeltaIsc. . . the jejunum. The results indicate that C7-sorbin stimulated NaCl neutral absorption and inhibited electrogenic Cl⁻ in rat and human intestinal epithelia. In addition, the antisecretory effect was essentially observed in the distal part of both rat and human intestine and the. . .

L3 ANSWER 3 OF 15 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1997:513570 CAPLUS
 DOCUMENT NUMBER: 127:158216
 TITLE: Anion channel-forming peptides and their use in treatment of diseases of epithelial transport
 INVENTOR(S): Tomich, John M.; Iwamoto, Takeo; Sullivan, Lawrence P.
 PATENT ASSIGNEE(S): Kansas State University Research Foundation, USA;

SOURCE:

University of Kansas Medical Center; Tomich, John M.;
Iwamoto, Takeo; Sullivan, Lawrence P.
PCT Int. Appl., 93 pp.

DOCUMENT TYPE:

CODEN: PIXXD2

LANGUAGE:

Patent
English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9726905	A1	19970731	WO 1997-US1103	19970127
W: AU, CA, JP, MX, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT,				
SE				
AU 9715829	A1	19970820	AU 1997-15829	19970127
US 6077826	A	20000620	US 1998-93227	19980608
PRIORITY APPLN. INFO.:				
			US 1996-591381	A 19960125
			US 1997-789155	A 19970124
			WO 1997-US1103	W 19970127

AB The present invention is directed to multiple-peptide channel assemblies for transport of anions such as chloride ions through epithelial cells, synthetic peptides capable of forming such channel assemblies and methods for using channel assemblies in therapeutic contexts for altering the flux of water across epithelial cells. The channel assemblies are composed of a plurality of peptides that assemble within the membrane and provide for alteration of the flux of water through the cell. The peptides are sol. in water to a level of at least 10 mM and exhibit at least about 50 % helical content when dispersed in a 40 % trifluoroethanol/60 % water soln.

The peptides ideally have the amino acid sequence ABC(X)_n.RTM.DEF, where A, B, C, D, F and X are individual amino acid residues, n ranges from 12-24 and at least one of the amino acids selected from the group consisting of A, B, and C is a charged amino acid, and at least one of the amino acids selected from the group consisting of D, E, and F is a charged

amino acid. The method hereof provides for altering flux of water from an epithelial cell and includes providing from 3-6 peptides capable of forming a channel assembly with each of such peptides having from about 18-30 amino acid residues therein, then contacting the peptides with a surface of an epithelial cell to cause the peptides to embed therein and alter the flux of water across the cell. Peptides derived from pore-forming domains of the strychnine-binding .alpha.-subunit of the inhibitory glycine receptor of human brain, the inhibitory GABA receptor of human brain, and the cystic fibrosis transmembrane conductance regulator of human epithelium were tested in MDCK cell culture and an animal model of cystic fibrosis. The peptides stimulated Cl⁻ transport and fluid secretion.

IT 7732-18-5, Water, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(alteration of flux across epithelial membranes of;
anion channel-forming peptides and their use in treatment of
diseases of epithelial transport)

L3 ANSWER 4 OF 15

MEDLINE

DUPLICATE 2

ACCESSION NUMBER: 1998057851 MEDLINE

DOCUMENT NUMBER: 98057851 PubMed ID: 9396069

TITLE: Comparison of the antisecretory effect of endogenous forms of peptide YY on fed and fasted rat jejunum.

AUTHOR: Eto B; Boisset M; Anini Y; Voisin T; Desjeux J F

CORPORATE SOURCE: Unite de Recherche sur les Fonctions Intestinales, le Metabolisme et la Nutrition, Institut National de la Sante

Saint-Lazare, et de la Recherche Medicale (INSERM), Hopital

Paris, France.. Biologie@cnam.fr
SOURCE: PEPTIDES, (1997) 18 (8) 1249-55.
Journal code: 8008690. ISSN: 0196-9781.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199801
ENTRY DATE: Entered STN: 19980217
Last Updated on STN: 19980217
Entered Medline: 19980130

AB It is intriguing that the antisecretory **peptide** YY is present in plasma in two forms: PYY1-36 and PYY3-36. PYY3-36 has been found in human and rabbit blood within 30 min of the beginning of the meal, when the peak

of **water** and electrolyte secretion occurs in the duodeno-jejunum. The aim of this study was therefore to compare the antisecretory effect of. . . The variations in electrolyte secretion were assessed by measuring the variations in short-circuit current (delta Isc) and transepithelial isotopic chloride **fluxes** in jejunal mucosa isolated from fed and fasted animal, and mounted in Ussing Chambers. In fasted animals, $2 \times 10(-7)$. . . contrast, in fed animals, $2 \times 10(-7)$ M PYY3-36 did not trigger a significant response on Isc and

net chloride **flux**, while the response to PYY1-36 was present but blunted. The absence of response was probably not related to the presence of secretory **peptides** because PYY3-36 was still able to induce a reduction in Isc after stimulation by a series of 10 different secretory **peptides**. After $10(-8)$ M PYY3-36 addition to an **epithelium** from the fasted animal, response to $10(-7)$ M PYY3-36 was blunted for 30 min and returned to control value after. . . but lack of activity in fed animals. These results suggest that the two circulating forms of PYY act as antisecretory **peptides** by two different mechanisms, implying a C-terminal specificity.

L3 ANSWER 5 OF 15 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 96116333 MEDLINE
DOCUMENT NUMBER: 96116333 PubMed ID: 8801342
TITLE: Different properties of the paracellular pathway account for the regional small intestinal permeability to the peptide desmopressin.
AUTHOR: Pantzar N; Lundin S; Westrom B R
CORPORATE SOURCE: Department of Animal Physiology, University of Lund, Sweden.
SOURCE: JOURNAL OF PHARMACEUTICAL SCIENCES, (1995 Oct) 84 (10) 1245-8.
Journal code: 2985195R. ISSN: 0022-3549.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199609
ENTRY DATE: Entered STN: 19961015
Last Updated on STN: 19961015
Entered Medline: 19960930

AB . . . vasopressin analogue desmopressin (dDAVP) was further characterized in proximal jejunal and distal (ileocecal) segments of the rat. Administration of the **peptide** to closed small intestinal loops confirmed the existence of regional absorption differences also in vivo in rats. Thus, the extent. . . ileocecal permeability could either be due to the presence of more permeable or dynamic pores in this region, where the **epithelial** surface area is smaller, or to an increased capacity for paracellular **water flux**. These results

may have relevance for drug transport in the small intestine, where site-specific delivery of drug or enhancing agents.

L3 ANSWER 6 OF 15 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 93354008 MEDLINE
DOCUMENT NUMBER: 93354008 PubMed ID: 8350667
TITLE: Atrial natriuretic factor enhances the hydroosmotic response of toad bladder to submaximal doses of vasopressin.
AUTHOR: Yu L; Tolvo A J; Scott W N
CORPORATE SOURCE: Department of Biology, New York University, NY 10003.
SOURCE: LIFE SCIENCES, (1993) 53 (7) 541-6.
Journal code: 0375521. ISSN: 0024-3205.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199309
ENTRY DATE: Entered STN: 19931001
Last Updated on STN: 19931001
Entered Medline: 19930910

AB Using the toad urinary bladder, we examined the effects of submaximal levels of atrial natriuretic factor (ANF) upon the hydroosmotic **water flux** caused by physiologic concentrations of vasopressin (VP). Pretreatment with ANF prior to the addition of VP (10(-9)M) significantly enhanced **water** transport (123 +/- 23%) compared to tissues exposed to VP alone. Pre-treatment with ANF also significantly enhanced the hydroosmotic response. . . . monophosphate (cyclic-AMP). When the concentration of VP was progressively increased during time course experiments, an inhibitory effect of ANF on **water** transport followed the early stimulatory response to this **peptide**. The data support a novel, dose-dependent modulatory role for ANF early in the response of transporting **epithelia** to VP. Moreover, the stimulatory effect of submaximal doses of ANF to cyclic-AMP mediated **water** transport suggest the possibility that modulation by ANF may occur at a site following the VP receptor-linked adenylate cyclase system.

L3 ANSWER 7 OF 15 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 92299677 MEDLINE
DOCUMENT NUMBER: 92299677 PubMed ID: 1318883
TITLE: Mechanism of cholera toxin action on a polarized human intestinal epithelial cell line: role of vesicular traffic.
AUTHOR: Lencer W I; Delp C; Neutra M R; Madara J L
CORPORATE SOURCE: Combined Program in Pediatric Gastroenterology and Nutrition, Children's Hospital, Boston, Massachusetts.
CONTRACT NUMBER: DK21505 (NIDDK)
DK35932 (NIDDK)
HD17557 (NICHD)
+
SOURCE: JOURNAL OF CELL BIOLOGY, (1992 Jun) 117 (6) 1197-1209.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199207
ENTRY DATE: Entered STN: 19920731
Last Updated on STN: 19970203
Entered Medline: 19920717

AB The massive secretion of salt and **water** in cholera-induced diarrhea involves binding of cholera toxin (CT) to ganglioside GM1 in the apical membrane of intestinal **epithelial** cells, translocation of the enzymatically active A1-**peptide** across the membrane, and subsequent activation of adenylate cyclase located on the cytoplasmic

surface of the basolateral membrane. Studies on . . . the hypothesis that toxin action in polarized cells may involve intracellular movement of toxin-containing membranes, monolayers of the polarized intestinal **epithelial** cell line T84 were mounted in modified Ussing chambers and the response to CT was examined. Apical CT at 37 . . . effective dose, ED50 integral of 0.5 nM) after a lag of 33 +/- 2 min which bidirectional 22Na+ and 36Cl- flux studies showed to be due to electrogenic Cl- secretion. The time course of the CT-induced Isc response paralleled the time. . . (30-50%) than the response to basolateral CT, even though translocation occurred in both cases as evidenced by the formation of A1-peptide. A functional rhodamine-labeled CT-analogue applied apically or basolaterally at 20 degrees C was visualized only within endocytic vesicles close to. . . movement into deeper apical structures was detected at 37 degrees C. At 15 degrees C, in contrast, reduction to the A1-peptide was completely inhibited and both apical and basolateral CT failed to stimulate Isc although Isc responses to 1 nM vasoactive intestinal **peptide**, 10 microM forskolin, and 3 mM 8Br-cAMP were intact. Re-warming above 32 degrees C restored CT-induced Isc. Preincubating monolayers for. . . response to apical toxin remained completely inhibited. These results identify a temperature-sensitive step essential to apical toxin action on polarized **epithelial** cells. We suggest that this event involves vesicular transport of toxin-containing membranes beyond the apical endosomal compartment.

L3 ANSWER 8 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 6

ACCESSION NUMBER: 1992:345325 BIOSIS
DOCUMENT NUMBER: BA94:37550
TITLE: SODIUM POTASSIUM CHLORIDE COTRANSPORT IN THE SHARK RECTAL GLAND I. REGULATION IN THE INTACT PERFUSED GLAND.
AUTHOR(S): FORBUSH B III; HAAS M; LYTLE C
CORPORATE SOURCE: DEP. CELLULAR MOLECULAR PHYSIOLOGY, YALE UNIV. SCH. MED., 333 CEDAR ST., NEW HAVEN, CONN. 06510.
SOURCE: AM J PHYSIOL, (1992) 262 (4 PART 1), C1000-C1008.
CODEN: AJPHAP. ISSN: 0002-9513.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB. . . intact gland. Glands were perfused with a shark Ringer solution, either in a basal state or stimulated with vasoactive intestinal **peptide** (VIP). [3H]benzmetanide was added to the perfusion solution for the last 25 min of perfusion, after which the gland was. . . play a dual role in the shark rectal gland: participation in secretagogue-stimulated net salt secretion and in the regulation of **epithelial** cell volume in the face of large transcellular salt and water fluxes.

L3 ANSWER 9 OF 15 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 92228703 MEDLINE
DOCUMENT NUMBER: 92228703 PubMed ID: 1808606
TITLE: The influence of peptide structure on transport across Caco-2 cells.
COMMENT: Comment in: Pharm Res. 1993 Apr;10(4):635-7
AUTHOR: Conradi R A; Hilgers A R; Ho N F; Burton P S
CORPORATE SOURCE: Drug Delivery Systems Research, Upjohn Company, Kalamazoo, Michigan 49001.
SOURCE: PHARMACEUTICAL RESEARCH, (1991 Dec) 8 (12) 1453-60.
Journal code: 8406521. ISSN: 0724-8741.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199205

ENTRY DATE: Entered STN: 19920607
Last Updated on STN: 19950206
Entered Medline: 19920519

AB The relationship between structure and permeability of **peptides** across **epithelial** cells was studied. Using confluent monolayers of Caco-2 cells as a model of the intestinal **epithelium**, permeability coefficients were obtained from the steady-state **flux** of a series of neutral and zwitterionic **peptides** prepared from D-phenylalanine and glycine. Although these **peptides** ranged in lipophilicity (log octanol/water partition coefficient) from -2.2 to +2.8, no correlation was found between the observed **flux** and the apparent lipophilicity. However, a strong correlation was found for the **flux** of the neutral series and the total number of hydrogen bonds the **peptide** could potentially make with **water**. These results suggest that a major impediment to **peptide** passive absorption is the energy required to break **water-peptide** hydrogen bonds in order for the solute to enter the cell membrane. This energy appears not to be offset by. . .

L3 ANSWER 10 OF 15 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 92179471 MEDLINE
DOCUMENT NUMBER: 92179471 PubMed ID: 1665571
TITLE: The effect of atrial natriuretic peptide on intestinal electrolyte transport.
AUTHOR: Catto-Smith A G; Hardin J A; Patrick M K; O'Loughlin E V; Gall D G
CORPORATE SOURCE: Intestinal Disease Research Unit, University of Calgary, Alberta, Canada.
SOURCE: REGULATORY PEPTIDES, (1991 Oct 1) 36 (1) 29-44.
Journal code: 8100479. ISSN: 0167-0115.
PUB. COUNTRY: Netherlands
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199204
ENTRY DATE: Entered STN: 19920424
Last Updated on STN: 19920424
Entered Medline: 19920407

AB The effect of atrial natriuretic **peptide** (ANP) on rat small intestinal electrolyte transport was examined. In vivo, intravenous administration of rat ANP(99-126) induced diuresis and natriuresis in conjunction with a significant decrease in intestinal **water** (basal, 37.1 +/- 5.7 versus ANP 28.5 +/- 6.0 microliters/cm per 20 min, P less than 0.05) and Na+ (4.0. . . in short circuit current and stimulated net Cl- secretion due to a significant increase in the unidirectional serosal to mucosal **flux** (JCl-sm: jejunum 17.4 +/- 1.3 versus 19.8 +/- 1.3 microEq/cm2 per h, P less than 0.01, n = 6; ileum.
. . . 5-HT2 receptor antagonist cinanserin (72 +/- 44%, P less than 0.05).
Guanylate cyclase activity was stimulated by ANP in intact **epithelium**, but not in isolated crypt and villus enterocytes.

L3 ANSWER 11 OF 15 MEDLINE DUPLICATE 9
ACCESSION NUMBER: 89255935 MEDLINE
DOCUMENT NUMBER: 89255935 PubMed ID: 2723060
TITLE: Immune system control of rat and rabbit colonic electrolyte transport. Role of prostaglandins and enteric nervous system.
AUTHOR: Bern M J; Sturbaum C W; Karayalcin S S; Berschneider H M; Wachsmann J T; Powell D W
CORPORATE SOURCE: Department of Medicine, University of North Carolina, Chapel Hill 27599.
CONTRACT NUMBER: DK-07463 (NIDDK)
DK-15350 (NIDDK)

DK-34987 (NIDDK)
SOURCE: JOURNAL OF CLINICAL INVESTIGATION, (1989 Jun) 83 (6)
1810-20.
Journal code: 7802877. ISSN: 0021-9738.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198907
ENTRY DATE: Entered STN: 19900306
Last Updated on STN: 19980206
Entered Medline: 19890712

AB . . . in controlling intestinal electrolyte transport was studied in rat and rabbit colon in Ussing chambers. A phagocyte stimulus, the chemotactic **peptide** FMLP, and a mast cell stimulus, sheep anti-rat IgE, caused a brief (less than 10 min) increase in short-circuit current. . . . activation, platelet-activating factor (PAF) and reactive oxygen species (ROS), caused a sustained, biphasic increase in the I_{sc}. Ion replacement and **flux** studies indicated that these agonists stimulated electrogenic Cl secretion and inhibited neutral NaCl absorption; responses that were variably inhibited by. . . had no effect on immune agonist-stimulated production of PGE₂ or PGI₂. These results indicate that immune system agonists alter intestinal **epithelial** electrolyte transport through release of cyclooxygenase products from cells in the lamina propria with at least 50% of the response. . . . The immune system, like the enteric nervous system and the endocrine system, may be a major regulating system for intestinal **water** and electrolyte transport in health and disease.

L3 ANSWER 12 OF 15 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 87118461 EMBASE
DOCUMENT NUMBER: 1987118461
TITLE: Natriuretic hormones: Comparison of renal effects.
AUTHOR: Bourgoignie J.J.
CORPORATE SOURCE: Division of Nephrology, Department of Medicine, University of Miami School of Medicine, Miami, FL 33101, United States
SOURCE: Klinische Wochenschrift, (1987) 65/SUPPL. 8 (14-20).
CODEN: KLWOAZ
COUNTRY: Germany
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
006 Internal Medicine
028 Urology and Nephrology
003 Endocrinology
LANGUAGE: English

AB . . . compounds are reviewed. One may originate from the hypothalamus (natriuretic factor-NF) and the other from the cardiac atria (atrial natriuretic **peptide**-ANP). Differences in the intrarenal mechanisms of action of NF and ANP should be anticipated in view of the fundamentally different. . . kidneys and in kidneys with a decreased functional mass. Effects on potassium excretion are variable. For both, the increases in **water** and solute excretion are associated with several sites of action within the nephron. Whereas NF has little effect on glomerular. . . and filtration fraction, ANP may induce impressive changes in kidney and in superficial nephron GFR, increasing the filtered load of **water** and solutes. ANP also increases filtration fraction changing, thereby, the peritubular physical forces of filtrate reabsorption. Both NF and ANP inhibit **water** and solute reabsorption in the proximal tubule. With NF, a direct tubular effect has been demonstrated in recollection micropuncture studies. In contrast, a direct **epithelial** effect has not been elicited with ANP in the proximal tubule. Nevertheless, the **peptide** markedly increased the fractional excretion of phosphate and lithium indicating a proximal tubular action, possibly hemodynamically mediated. Neither compound has. . . in the medullary segment with ANP. In the cortical collecting

tubule, NF inhibits net sodium reabsorption by inhibiting the unidirectional **flux** of sodium from lumen to peritubular capillary. In the medullary collecting duct, ANP increases tubular fluid sodium concentration and delivery. . . in blood flow and pressures, medullary interstitial washout and changes in the sodium permeability of the inner medullary collecting duct **epithelium**.

L3 ANSWER 13 OF 15 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 85217648 EMBASE

DOCUMENT NUMBER: 1985217648

TITLE: Localization and properties of angiotensin receptors.

AUTHOR: Mendelsohn F.A.O.

CORPORATE SOURCE: University of Melbourne, Department of Medicine, Austin Hospital, Heidelberg 3084, Vic., Australia

SOURCE: Journal of Hypertension, (1985) 3/4 (307-316).

CODEN: JOHYD3

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal

FILE SEGMENT: 037 Drug Literature Index

006 Internal Medicine

030 Pharmacology

018 Cardiovascular Diseases and Cardiovascular Surgery

003 Endocrinology

LANGUAGE: English

AB Angiotensin II (ANG II), the effector **peptide** of the renin-angiotensin system, exerts a wide variety of actions on the cardiovascular, renal, endocrine, metabolic and central and peripheral.

. such as stimulation of contractile properties of the myocardium, modulation of pituitary hormone release, increasing hepatic glucose metabolism, stimulation of **water** and electrolyte **flux** in a variety of **epithelia** and direct cellular actions of the **peptide** at the nuclear level have been demonstrated, but their physiological or pathophysiological significance is not yet clear. The localization and. . .

L3 ANSWER 14 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 1984:292819 BIOSIS

DOCUMENT NUMBER: BA78:29299

TITLE: ORIGINS OF PEPTIDE AND NOREPINEPHRINE NERVES IN THE MUCOSA OF THE GUINEA-PIG SMALL INTESTINE.

AUTHOR(S): KEAST J R; FURNESS J B; COSTA M

CORPORATE SOURCE: DEP. HUMAN MORPHOL., FLINDERS UNIV. SOUTH AUSTRALIA, BEDFORD PARK, S.A. 5042, SOUTH AUSTRALIA.

SOURCE: GASTROENTEROLOGY, (1984) 86 (4), 637-644.

CODEN: GASTAB. ISSN: 0016-5085.

FILE SEGMENT: BA; OLD

LANGUAGE: English

IT Miscellaneous Descriptors

MYENTERIC PLEXUS VASOACTIVE INTESTINAL **PEPTIDE** SUBSTANCE P
SOMATOSTATIN NEURO **PEPTIDE** Y CHOLECYSTOKININ ENKEPHALIN
GASTRIN RELEASING **PEPTIDE** 5 HYDROXY TRYPTAMINE ACETYL CHOLINE
TRANS **EPITHELIAL WATER FLUX** TRANS
EPITHELIAL ELECTROLYTE FLUX

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10

ACCESSION NUMBER: 1979:228009 BIOSIS

DOCUMENT NUMBER: BA68:30513

TITLE: CALCIUM IONOPHORE STIMULATED ION SECRETION IN RABBIT ILEAL MUCOSA RELATION TO ACTIONS OF CYCLIC AMP AND CARBAMYL CHOLINE.

AUTHOR(S): BOLTON J E; FIELD M

CORPORATE SOURCE: GASTROINTEST. UNIT, BETH ISR. HOSP., BOSTON, MASS. 02215, USA.

SOURCE: J MEMBR BIOL, (1977) 35 (2), 159-173.

CODEN: JMBBBO. ISSN: 0022-2631

FILE SEGMENT: BA; OLD

LANGUAGE: English

AB. . . current and resistance, net secretion of Cl due both to a decrease in the unidirectional mucosa (m) to serosa (s) flux and an increase in the (s) to (m) flux and net secretion of Na due to a decrease in (m) to (s) flux. Measurements of intracellular cAMP level demonstrated no change following incubation with the ionophore. Removal of Ca from the serosal bathing. . . the p.d. [potential difference] response to A23187. The ionophore apparently elicits its secretory actions by increasing Ca influx into the epithelial cells. Carbamylcholine and serotonin [5-hydroxytryptamine], secretagogues known to have no effect on intracellular cAMP level in intestinal mucosa, were dependent. . . full electrical response (although, in the case of carbamylcholine at least, Sr can substitute for Ca). The secretagogues vasoactive intestinal peptide and prostaglandin E1, which raise cAMP concentration in intestinal mucosa, do not appear to require external

Ca. Ca may be an intracellular mediator of intestinal ion and water secretion and some intestinal secretagogues may act as Ca ionophores.

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